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Predictors of Emotional Numbing in Posttraumatic Stress Disorder

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Little is known about the mechanisms underlying emotional numbing (EN). The functional relationship between other classes of posttraumatic stress disorder (PTSD) symptoms and EN is also not well understood. In the present study, we examined the statistical predictors of EN. We hypothesized that the severity of EN would be most strongly associated with the hyperarousal symptoms rather than the avoidance symptoms of PTSD, or comorbid depression or substance abuse. This prediction was derived from psychological and biological models that posit EN to be a product of the depletion of emotional resources subsequent to chronic hyperarousal. Using hierarchical multiple regression in two separate samples of Vietnam combat veterans, we found hyperarousal symptoms to be the most robust predictor of EN. These data suggest that there is a substantive relationship between hyperarousal symptoms and EN in PTSD.

KEY WORDS: emotional numbing.

Emotional numbing (EN) in posttraumatic stress disorder (PTSD) is a cluster of debilitating symptoms involving problems in the experience and expression of emotion. In the DSM-III-R and DSM-IV (American Psychiatric Association [APA], 1987, 1994) EN is represented in three separate diagnostic criteria: markedly diminished interest in significant activities (Criterion C-4), feelings of detachment or estrangement from others (C-5),

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and restricted range of affect (C-6). Although seen as a hallmark of PTSD by many clinical researchers (e.g., van der Kolk, Greenberg, & Boyd, 1985), little is known about the mechanisms underlying EN, and the functional relationship between other symptoms of PTSD and EN is not well understood (Davidson & Foa, 1992; Litz, 1992). The extent to which reports of EN are associated with other psychiatric problems, such as major depression or substance abuse, has also been unspecified. In addition, until recently, it has been unclear whether EN is indeed a distinct construct within the PTSD syndrome.

In the DSM-III-R and DSM-IV, EN symptoms are grouped, under Category C, with the two avoidance-related symptoms of PTSD—avoidance of thoughts and feelings related to the trauma (C-1) and avoidance of external reminders of the trauma (C-2)—suggesting that EN is part of a larger class of avoidance symptoms. Recent factor analytic studies of PTSD symptoms, however, have shown that the three EN symptoms form a distinct factor, separate from both the two avoidance symptoms and from the reexperiencing (Criterion B) symptoms and hyperarousal (Criterion D) symptoms (Foa, Riggs, & Gershuny, 1995; King & King, 1994; Weathers & Litz, 1993). Furthermore, when examined separately from the avoidance symptoms the three EN symptoms independently contribute to the prediction of both the diagnosis of PTSD and poor psychosocial outcome in traumatized populations (Breslau & Davis, 1992; Foa et al., 1995; Kilpatrick & Resnick, 1993).

There are several possible explanations for the "numbness" or lack of emotional responsiveness that some PTSD patients report. First, EN symptoms may result from chronic avoidance of environmental and experiential reminders of the trauma, which could reduce exposure to emotion-eliciting situations or serve to suppress the internal experience of emotion (see Keane, Fairbank, Caddell, Zimering, & Bender, 1985). Second, EN may be secondary to the biological sequelae of traumatization and conditioned fear including the depletion of catecholamines (van der Kolk et al., 1985), and/or a conditioned analgesic effect that is caused by the release of endogenous opioids during conditioned fear responses that serve to suppress pain and tranquilize the organism (Pitman, van der Kolk, Orr, & Greenberg, 1990). Relatedly, from a psychological perspective, PTSD patients may expend so much cognitive, behavioral, and emotional effort attempting to manage their hyperarousal and reactivity symptoms that they exhaust or deplete their emotional resources, leading to a lack of responsiveness to subsequent appetitive stimulation and a decrease in hedonic capacity (e.g., Foa, Zinbarg, & Rothbaum, 1992; Litz, 1992). Third, EN may reflect symptoms of a comorbid disorder. For example, diminished interest (C-4) may be the same phenomenon as anhedonia in depression. Similarly, EN may

be a biological or psychological sequela of chronic substance abuse that is often comorbid with PTSD (e.g., Kulka et al., 1990).

The present study is an empirical investigation of the EN symptoms aimed at improving our understanding of their role in the PTSD syndrome. In order to provide a preliminary test of each of the above theories, we used hierarchical multiple regression to determine the extent to which EN symptoms are predicted by other aspects of the PTSD syndrome, depression, or substance abuse.

We predicted that, after controlling for race, age, extent of war-zone exposure, depression, substance abuse, and PTSD symptom clusters, the best predictor of EN would be the extent of hyperarousal symptoms (Criterion D). The first three variables are included because they have been associated with differential risk for PTSD (e.g, Kulka et al., 1990). This prediction was based on conceptual models suggesting that rather than simply being explained by avoidance, or related to depression or substance abuse, EN-related problems in PTSD are considered to be manifestations of the deficits that arise from the depletion of biological, cognitive and affective resources secondary to chronic hyperarousal in traumatized individuals (e.g., Barlow, 1988; Foa et al., 1992; Litz, 1992).

Method

Participants and Procedure

Two separate groups of Vietnam combat veterans were studied. The first was a large, community-based probability sample of veterans comprehensively assessed for PTSD in the Clinical Examination Component of the National Vietnam Veterans Readjustment Study (NVVRS; Kulka et al., 1990). The second group, used to replicate and extend the findings from the first sample, was a group of service-seeking Vietnam veterans assessed at the National Center for PTSD at the Boston Department of Veterans Affairs Medical Center.

NVVRS Clinical Examination Subsample. The Clinical Examination Subsample of the NVVRS is a stratified probability subset of participants from the larger, nationally representative survey sample of the NVVRS, labeled the National Survey of the Vietnam Generation (NSVG; see Kulka et al. [1990] and Schlenger et al. [1992] for a detailed account of the NVVRS study methods). The NSVG entailed structured interviews by survey interviewers on 1,632 men and women veterans who served in the Vietnam theater of operations. The Clinical Examination Subsample was a stratified subsample (N = 334 theater veterans) selected from the NSVG

to receive a full psychodiagnostic interview by doctoral-level experts in the field of PTSD. Participants in the Clinical Examination Subsample underwent a thorough clinical assessment that consisted of multiple PTSD measures, including a formal diagnostic interview using the Structured Clinical Interview for DSM-III-R (SCID; Spitzer, Williams, Gibbon, & First, 1990).

Boston sample. Participants were 123 service-seeking Vietnam theater veterans seen at the National Center for PTSD at the Boston Department of Veterans Affairs Medical Center. Veterans underwent a comprehensive psychodiagnostic evaluation in the context of a clinical research project. Participants were interviewed by doctoral-level clinicians who administered both the full SCID and the Clinician-Administered PTSD Scale (CAPS; Blake et al., 1990). The CAPS is a structured clinical interview that evaluates both the frequency and the intensity of the 17 symptoms of PTSD. It has been shown to have excellent reliability and diagnostic utility (Weathers & Litz, 1994). Participants were also administered the Combat Exposure Scale (Keane et al., 1989).

Data Analysis Plan

We conducted a series of hierarchical multiple regression analyses to examine the separate and combined contributors to the variance of EN. The DSM-III-R/DSM-IV PTSD symptoms of diminished interest (C-4), detachment (C-5), and restricted range of affect (C-6) were combined into a composite index, which was used as the dependent measure for each model. We were able to employ multiple methods of deriving the EN (dependent) variable as well as the other PTSD symptom clusters employed as independent variables, because two distinct PTSD indicators were employed in the Boston sample. The result was a reduction in mono-method bias, thus enhancing the validity of our findings (Cook & Campbell, 1979). We sought to replicate the findings from the community sample with a clinical sample, and to replicate the analysis across different indices of the constructs of interest.

All independent variables were entered hierarchically in each model, with hyperarousal symptoms (Category D) always entered last, providing the most conservative test of the relationship between EN and hyperarousal symptoms. In each instance, a significance level of .05 was used as the criterion for inclusion. Conceptually, the independent variables selected as predictors of the EN dependent variables were consistent in the NVVRS and Boston samples. However, there were several differences between these variables that were determined by the unique assessment methods used in the two studies, as described below.

The following were used as independent measures in the NVVRS analysis (listed in order of entry): (a) age, gender, race (defined as Caucasian or not), (b) mean score on the war-zone exposure scale (Kulka et al., 1990), (c) the presence or absence of current SCID-diagnosed major depression, (d) the presence or absence of current SCID-diagnosed substance abuse or dependence, (e) the sum of the number of current SCID-endorsed Criterion B symptoms (0-4), (f) the sum of the number of current SCID-endorsed PTSD avoidance symptoms (0-2), and (g) the sum of the number of current SCID-endorsed Criterion D symptoms (0-6). The dependent variable that represented EN in the above model was a composite sum of the number of current SCID endorsed EN symptoms (ranging from 0-3).

Three separate models were used to examine the predictors of EN in the Boston sample. In each model, age and race were entered first as a block, followed by Combat Exposure Scale scores. This was followed by the presence or absence of current major depression and current substance abuse or dependence, as assessed by the SCID. This was followed by the non-EN PTSD symptom clusters, each entered as a block in the following order: re-experiencing symptoms (Category B), avoidance symptoms (Symptoms C-1 and C-2), and hyperarousal symptoms (Category D). Gender was not included as a predictor in the Boston sample since there were no women veterans in that sample.

The three dependent variables representing EN in the Boston data set were: (a) the sum of the number of SCID-endorsed EN symptoms (identical to the variable used in the NVVRS sample), (b) the mean severity rating (frequency plus intensity) across the three CAPS EN symptoms, and (c) the composite combination of scores from each index of EN, computed by deriving the mean of the *z*-scores of the number of SCID-defined EN symptoms and the mean CAPS EN symptoms. The composite index was used as a dependent measure in order to provide a more reliable index of EN. Depending on the model, the non-EN PTSD independent variables were defined as the sum of the number of SCID items endorsed, the mean severity of the relevant CAPS items, or the *z*-score composite mean of the SCID and CAPS-defined variables.

The three regression equations employed with the Boston dataset can be schematically outlined in the following manner: (a) SCID-defined EN = all other SCID-defined (non-EN) PTSD variables (this model directly replicated the model employed with the NVVRS data), (b) CAPS-defined EN = all other CAPS-defined (non-EN) PTSD variables, and (e) the *z*-score composite SCID/CAPS-defined EN = all other *z*-score composite SCID/CAPS (non-EN) PTSD variables.

Results

Table 1 shows the demographic characteristics of the NVVRS and the Boston samples. The samples were comparable in age, but the Boston sample was less well-educated, had fewer Hispanics, and had fewer veterans who were currently married. Table 1 also shows the PTSD prevalence rates for the two samples (based on the SCID-PTSD module). As would be expected, the Boston sample had more than twice the prevalence rate of PTSD.

Table 2 shows the findings from all the multiple regression analyses of EN symptoms. As predicted, the number of hyperarousal (Criterion D) symptoms was the best predictor of EN using the NVVRS data. The standardized regression coefficient (beta) for the D-symptoms, reflecting the relative contribution of the dependent measure was high (.58), as was the partial correlation (.56). The composite of the avoidance symptoms, C-1 and C-2, was also a significant predictor, but the relative contribution was

Table 1. Demographic and PTSD Prevalence Characteristics of the Two Vietnam Combat Veteran Samples[a]

Variable	Boston (All men) (N = 123)	NVVRS Men (n = 259)	NVVRS Women (n = 84)
Age			
M (*SD*)	43.7 (2.7)	42.3 (2.7)	45.1 (3.2)
Race			
Caucasian	74.4%	39.0%	91.7%
African-American	23.1%	28.2%	2.4%
Hispanic	2.5%	32.8%	6.0%
Marital status			
Married	28.7%	81.1%	52.4%
Separated	13.9%	3.5%	2.4%
Divorced	27.9%	10.0%	9.5%
Single	26.2%	5.0%	33.3%
Widowed	2.5%	0.4%	2.4%
Educational level			
Some high school	10.7%	7.3%	0.0%
High school/GED	24.8%	26.3%	11.9%
Some college	35.5%	50.2%	31.0%
College	7.4%	5.8%	22.6%
Grad./prof. school	7.4%	10.4%	34.5%
PTSD Positive Cases	55.3%	25.5%	10.7%

[a]PTSD cases were identified by SCID-based diagnosis in both datasets.

Table 2. Significant Predictors of Emotional Numbing Symptoms[a]

	SCID			CAPS			SCID/CAPS		
		C-4,5,6 Composite Sum			C-4,5,6 Composite Mean			C-4,5,6 Composite Mean *z*	
Model/Predictor	beta	*p*	partial *r*	beta	*p*	partial *r*	beta	*p*	partial *r*
NVVRS sample—SCID									
Sum of SCID C-1,2	.19	.0001	.21		—			—	
Sum of SCID D	.58	.0001	.56		—			—	
Boston sample—SCID									
SCID-depression	.16	.02	.33		—			—	
Sum of SCID C-1,2	.22	.007	.25		—			—	
Sum of SCID-D	.39	.004	.22		—			—	
Boston sample—CAPS									
SCID-Depression		—		.24	.0003	.34		—	
Mean CAPS D Severity		—		.55	.0001	.43		—	
Boston sample—CAPS/SCID composite									
SCID-Depression		—			—		.17	.002	.10
Mean CAPS D Severity		—			—		.69	.0001	.54

[a]The model statistics for the equation employing the NVVRS sample are: $F = 161.0$, $p = .0001$, Adjusted $R^2 = .50$. The model statistics for the equation employing the Boston sample, using the SCID are: $F = 20.54$, $p = .0001$, Adjusted $R^2 = .50$. The model statistics for the equation employing the Boston sample, using the CAPS are: $F = 21.66$, $p = .0000$, Adjusted $R^2 = .59$. The model statistics for the equation employing the Boston sample, using the CAPS/SCID composite are: $F = 36.55$, $p = .0000$, Adjusted $R^2 = .71$.

smaller (beta = .19, partial r = .21). No other independent variables were statistically significant predictors of EN in the equation employing the NVVRS data. The overall regression model was statistically significant and accounted for .50 of the variance in the reports of EN symptoms as defined by the SCID.

Table 2 also shows the results of the equation that was used to cross-validate the analysis of the NVVRS data using the Boston data. In this equation, Criterion D symptoms also emerged as a unique predictor of EN symptoms (beta = .39, partial r = .33). As in the NVVRS data set, the avoidance variable was also a significant predictor of EN in the Boston data (beta = .22, partial r = .25). Unlike the findings from the community sample, the SCID diagnosis of major depression was a significant predictor of EN in the Boston sample (beta = .16, partial r = .22).

The two additional multiple regression models predicting EN using the Boston data were also statistically significant and predicted a large percentage of the variance in each EN variable, as shown in Table 2. In each of the models that employed the CAPS, the SCID diagnosis of major depression was a significant predictor of EN. However, in each instance, although entered last, hyperarousal significantly accounted for unique variance in EN (ranging from 5% to 12%). In the model employing the CAPS variables, the beta for the mean of Criterion D symptoms was .55. In the model employing the z-score composite of the CAPS and the SCID variables, the beta for the mean z-score of the composite Criterion D symptoms was .69. No other independent variables in these models were statistically significant.

Discussion

The emotional numbing symptoms of PTSD (disinterest, detachment, and restricted range of affect) were consistently predicted by reports of hyperarousal symptoms in both a community-based probability sample and a service-seeking sample of Vietnam theater veterans. These correlational data suggest the possibility of a functional relationship between post-trauma arousal-related difficulties and constrictions of the experience and expression of emotion in trauma survivors. The generalizability of this conclusion is supported by the concordant findings across the two samples and across alternative measures of the constructs of interest.

The two avoidance symptoms of PTSD were also found to be a significant, though less robust, predictor of EN symptoms in both the community and clinical samples (when using the SCID as the source of PTSD data). This suggests that there may be a substantive relationship between

effortful avoidance and EN (Foa & Riggs, 1992). Alternatively, it could be that when patients report avoiding thoughts and feelings related to the trauma or having difficulty experiencing and expressing emotions on the SCID, they are reporting virtually the same problem.

Another finding of the present study was the absence of an association between a diagnosis of major depression and the EN-related symptoms in the community sample, suggesting that EN-related symptoms are not simply accounted for by comorbid major depression. In addition, we found no significant relationship between EN and the diagnosis of substance abuse in either sample. Taken together, these findings suggest that EN cannot be solely accounted for by the presence of these common comorbid disorders. In the clinical sample, however, the SCID diagnosis of major depression was a consistent predictor of EN. On the one hand this is not surprising given the consistently strong relationship between depression and PTSD in clinical populations (e.g., Orr et al., 1990). On the other hand there may be a substantive relationship between EN and symptoms of depression that surfaced due to the greater severity of the symptoms expressed in the clinical sample. Since only global indices of depression and substance abuse were used in the present study, it remains unclear whether specific clusters of symptoms within these problem areas bear a specific functional relationship with symptom reports of EN. Future research will need to better pinpoint the specific relationship between EN (and all PTSD symptom clusters for that matter) and other types of symptoms and disorders commonly reported by traumatized individuals.

Our findings provide correlational support for theories of PTSD suggesting that hyperarousal and reactivity lead to either a depletion of biological and psychological emotional-processing resources (e.g., Foa et al., 1992; Litz, 1992) or the activation of a defensive/inhibitory response (e.g., Horowitz, 1986). The converse, however, may also be true. That is, that EN-related psychological and biological processes can lead to greater hyperarousal problems. Indeed, there is a body of evidence demonstrating that acute and chronic suppression of emotional expression leads to greater physiological arousal disturbances (e.g., Gross & Levenson, 1993; Notarious & Levenson, 1979). In addition, there are some data to suggest that a low level of emotion-focused self-disclosure after a trauma causes greater hyperarousal and somatic problems (e.g., Pennebaker et al., 1989).

At this point, experimental studies are needed to examine the causal relationship between hyperarousal and EN-related symptoms. For example we are currently manipulating arousal-level in PTSD patients and measuring subsequent psychophysiological reactions to positively and negatively valenced emotional images, expecting a diminution in emotional response in hyperaroused states (Orsillo, Litz, Bergman, Block, & Kaloupek, 1995).

In addition, it would be interesting to reduce hyperarousal in PTSD patients by biochemical means (e.g., beta-blockade) or by relaxation methods, and then evaluate changes in emotional responsivity.

One of the methodological limitations of the present study is the nature of the data used to index EN-related problems. Our assessment procedures required participants to make highly general judgments about their emotional behavior. The relationship of global retrospective judgments to actual emotional behavior in various challenging situations has recently been called into question on both conceptual and methodological grounds (Litz, 1992). Thus far EN has been treated as a stable and enduring characteristic of PTSD in the literature which has precluded a critical examination of the limiting conditions and situational constraints (both environmental and internal to the person) of such problems. It is quite likely that, if asked, PTSD patients would report considerable variability in their emotional experience. For example, they may report feeling uninterested, disengaged, or unemotional some of the time, in certain situations, or when they are tense, anxious, and generally aroused. One of the implications of the present study is that it may be useful to begin to conceptualize and measure EN as depending, at least in part, on hyperarousal-related states in traumatized individuals.

In summary, using both community and service-seeking samples, we found the hyperarousal symptoms to be the most consistent statistical predictor of EN. Future research is needed to elucidate the causal relationship between hyperarousal and EN as well as the underlying processes that account for the observed correlation. In addition, our data revealed a relationship between depression and EN in the clinical sample but not in the community sample. Future research will need to disentangle the complex relationship between the phenomenology of depression and EN, as well as determine if in clinical samples, a third variable, such as general distress, accounts for the overlapping relationship.

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References

American Psychiatric Association. (1987). *The diagnostic and statistical manual of mental disorders (3rd ed., rev.)*. Washington, DC.: Author.

American Psychiatric Association. (1994). *The diagnostic and statistical manual of mental disorders (4th edition)*. Washington, DC: Author.

Barlow, D. H. (1988). *Anxiety and its disorders*. New York: The Guilford Press.

Blake, D. D., Weathers, F. W., Nagy, L. N., Kaloupek, D. G., Klauminzer, G., Charney, D. S., & Keane, T. M. (1990). A clinician rating scale for assessing current and lifetime PTSD: The CAPS-1. *The Behavior Therapist, 13*, 187-188.

Breslau, N., & Davis, G. C. (1992). Posttraumatic stress disorder in an urban population of young adults: Risk factors for chronicity. *American Journal of Psychiatry, 149*, 671-675.

Cook, T. D., & Campbell, D. T. (1979). *Quasi-experimentation: Design and analysis issues for field settings*. Boston: Houghton Mifflin.

Davidson, J., & Foa, E. (1991). Diagnostic issues in posttraumatic stress disorder: Considerations for the DSM-IV. *Journal of Abnormal Psychology, 100*(3), 346-355.

Foa, E. & Riggs, D. (1993). Posttraumatic stress disorder and rape. In J. M. Oldham, M. B. Riba, & A. Tasman (Eds.), *American Psychiatric Press review of psychiatry vol. 12*, Washington, DC.: American Psychiatric Press.

Foa, E., Riggs, D., & Gershuny, B. (1995). Arousal, numbing, and intrusion: Symptom structure of PTSD following assault. *American Journal of Psychiatry, 152*, 116-120.

Foa, E., Zinbarg, R., & Rothbaum, B. (1992). Uncontrollability and unpredictability in post-traumatic stress disorder: An animal model. *Psychological Bulletin, 112*, 218-238.

Gross, J. J., & Levenson, R. W. (1993). Emotional suppression: Physiology, self-report, and expressive behavior. *Journal of Personality and Social Psychology, 64*, 970-986.

Horowitz, M. (1986). *Stress response syndromes* (2nd ed.). Northvale, NJ: Jason Aronson.

Keane, T. M., Fairbank, J. A., Caddell, J. M., Zimering, R. T., & Bender, M. E. (1985). A behavioral approach to assessing and treating post-traumatic stress disorder in Vietnam veterans. In C. R. Figley (Ed.), *Trauma and its wake* (pp. 257-294). New York: Brunner/Mazel.

Keane, T. M., Fairbank, J. A., Caddell, J. M., Zimering, R. T., Taylor, K. L., & Mora, C. A. (1989). Clinical evaluation of a measure to assess combat exposure. *Psychological Assessment: A Journal of Consulting and Clinical Psychology, 1*, 53-55.

Kilpatrick, D., & Resnick, H. (1993). Posttraumatic stress disorder associated with exposure to criminal victimization in clinical and community populations. In J. Davidson & E. Foa (Eds.) *Posttraumatic stress disorder: DSM-IV and beyond* (pp. 113-143). Washington, DC: American Psychiatric Press.

King, L. A., & King, D. W. (1994). Latent structure of the Mississippi Scale for Combat-related Posttraumatic Stress Disorder: Exploratory and higher-order confirmatory factor analyses. *Assessment, 1*, 275-291.

Kulka, R., Schlenger, W., Fairbank, J., Hough, R., Jordan, B., Marmar, C., & Weiss, D. (1990). *Trauma and the Vietnam war generation*. New York: Brunner Mazel.

Litz, B. (1992). Emotional numbing in combat-related post-traumatic stress disorder: A critical review and reformulation. *Clinical Psychology Review, 12*, 417-432.

Notarious, C. I., & Levenson, R. J. (1979). Expressive tendencies and physiological response to stress. *Journal of Personality and Social Psychology, 37*, 1204-1210.

Orr, S., Claiborn, J., Altman, B., Forgue, D., de Jong, J., Pitman, R., & Herz, L. (1990). Psychometric profiles of post-traumatic stress disorder, anxious, and healthy Vietnam veterans: Correlations with psychophysiologic responses. *Journal of Consulting and Clinical Psychology, 58*, 329-335.

Orsillo, S. M., Litz, B. T., Bergman, E. D., Block, D. & Kaloupek, D. G. (1995, November). *The suppression of emotional expression subsequent to conditioned emotional reactivity in PTSD*. Paper presented at the meeting of the Association for Advancement of Behavior Therapy, Washington D.C.

Pennebaker, J., Barger, S., & Tiebout, J. (1989). Disclosure of traumas and health among Holocaust survivors. *Psychosomatic Medicine, 51,* 577-589.

Rothbaum, B. O., Foa, E. B., Riggs, D. S., Murdock, T., & Walsh, W. (1992). A prospective examination of posttraumatic stress disorder in rape victims. *Journal of Traumatic Stress, 5,* 455-476.

Schlenger, W., Kulka, R., Fairbank, J., Hough, R., Jordan, B., Marmar, C., & Weiss, D. (1992). The prevalence of posttraumatic stress disorder in the Vietnam generation: A multimethod, multisource assessment of psychatric disorder. *Journal of Traumatic Stress, 5,* 333-363.

van der Kolk, B., Greenberg, M., Boyd, H., & Krystal, J. (1985). Inescapable shock, neurotransmitters, and addiction to trauma: Toward a psychobiology of post-traumatic stress. *Biological Psychiatry, 20,* 314-325.

Weathers, F., & Litz, B. (1993, August). Symptom patterns in combat-related PTSD: Clinical versus community samples. In B. T. Litz, Symposium Chair, *Manifestations of PTSD in traumatized populations—A symptom-level analysis.* Paper presented at the annual meeting of the American Psychological Association, Toronto.

Weathers, F. W., & Litz, B. T. (1994). Psychometric properties of the Clinician Administered PTSD scale. *PTSD Research Quarterly, 5,* 2-6.